Urban Sprawl and Risk for Being Overweight or Obese

Russ Lopez, MCRP, DSc

Objectives. I examined the association between urban sprawl and the risk for being overweight or obese among US adults.

Methods. A measure of urban sprawl in metropolitan areas was derived from the 2000 US Census; individual-level data were obtained from the Behavioral Risk Factor Surveillance System. I used multilevel analysis to assess the association between urban sprawl and obesity.

Results. After I controlled for gender, age, race/ethnicity, income, and education, for each 1-point rise in the urban sprawl index (0–100 scale), the risk for being overweight increased by 0.2% and the risk for being obese increased by 0.5%.

Conclusions. The current obesity epidemic has many causes, but there is an association between urban sprawl and obesity. (*Am J Public Health.* 2004;94:1574–1579)

Obesity is a health problem that rivals smoking in importance. Obese persons have an increased risk for diabetes,[1] cardiovascular disease,[2] cancer,[3] and mortality.[4,5] Obesity and overweight rates are increasing rapidly in the United States.[6,7] In 2000, approximately 20.1% of the adult population was obese and 36.7% was overweight. Similarly, the current National Health and Nutrition Examination Survey (1999–present) found that the percentage of obese adults increased from 22.9% between 1988 and 1994 to 30.5% between 1999 and 2000.[8] Childhood obesity rates also increased between 1988 to 1994 and 1999 to 2000 from 7.2% to 10.4% among children aged 2 to 5 years.[9] The southern states were the first to have more than 20% of their adult populations obese, and from this center, higher rates of obese and overweight adults have spread to all areas of the country.[10,11] Many of the metropolitan areas that have the highest levels of urban sprawl are located in the South. This association was 1 of the first links between levels of urban sprawl and the risk for being obese or overweight.

Urban sprawl is often loosely defined, and complicating these definitions is confusion among causes, consequences, and attributes of urban sprawl. For this study, urban sprawl was defined as an overall pattern of development across a metropolitan area where large percentages of the population live in lower-density residential areas. The causes of urban sprawl are not well identified but include affluence that enables households to purchase larger houses on larger lots, cultural values that reject urban living and emphasize automobile use, inexpensive land values that support urban sprawl–dependent lifestyles, and government policies that promote urban sprawl.[12–14] The consequences of urban sprawl include increased reliance on automobile transportation and decreased ability to walk to destinations, decreased neighborhood cohesion, and environmental degradation (e.g., greenhouse gas emissions and destruction of open space).[15–18]

Environmental factors also may contribute to obesity. Environments rich in sources of caloric food, poor street patterns, lack of pedestrian amenities, difficult-to-access destinations, and neighborhood perceptions all have been hypothesized to contribute to decreasing physical activity and to promote the development of obesity.[19–23] The Centers for Disease Control and Prevention (CDC) released a report that connected urban sprawl and obesity.[24] Others also have concluded that urban sprawl contributes to obesity, but they have not provided factual evidence to support these claims.[25,26] However, others maintain that urban sprawl is not associated with obesity and argue that affluence and lower-population densities encourage physical activity.[27] I examined potential associations between urban sprawl and the risk for being overweight or obese to determine if urban sprawl is a public health problem.

METHODS

This is a multilevel study, because it combines individual-level variables with 1 higher-level variable—urban sprawl—that is measured at the metropolitan level. Multilevel analysis is an appropriate means for assessing the association between individual- and community-level risk factors.[28–30] I used data from the 2000 Behavioral Risk Factor Surveillance System (BRFSS). The BRFSS is an annual telephone survey of adults, and there were 184 450 participants in 2000. The survey has more than 200 self-reported and calculated variables and is a good source of information about the health status and habits of the US population. Within each state, blocks of 100 telephone numbers constitute a primary sampling unit, and the goal is to interview 3 households within each primary sampling unit. The data are weighted on the basis of the probability of the household's telephone number being selected and the number of adults and telephones within the household. The sample also is adjusted for nonresponses and for households without telephones. Finally, there is an adjustment to ensure the sample contains representative proportions of selected demographic characteristics (gender, age, and race/ethnicity).[31]

The BRFSS excludes institutionalized persons, and response rates vary by state (range was 44%–95% in 1999). Data from the 2000 survey were obtained from the BRFSS Web site.[32] Respondents were assigned a sprawl index value for their metropolitan area on the basis of the metropolitan-area identifier in the survey. Respondents who lived in Puerto Rico (approximately 5% of the total survey), who lived outside metropolitan areas (approximately 30%), and who lived in metropolitan areas not identified (approximately 10%) were excluded from this study.

Obesity

Obese or overweight status is usually determined by the body mass index (BMI) formula (weight in kilograms divided by height in meters squared); adults are considered overweight when their BMI is greater than 25 and obese when their BMI is greater than 30.[33–34] BMI was calculated with respondents' self-reported heights and weights.

Urban Sprawl

Researchers at the Boston University School of Public Health used the 2000 US Census to develop an index that measured urban sprawl on the basis of density and compactness. It is important to note that sprawl is more than density, although density is a central component. Urban sprawl also is a function of how density is distributed across a metropolitan area. The federal Office of Management and Budget produced geographic definitions of all US metropolitan areas that consisted of 1 or more central cities and their surrounding counties. The US Bureau of the Census divided the country into tracts of approximately 4000 persons; beginning in 1990, the bureau used Geographic Information Systems to estimate the land area of tracts, which enabled the calculation of tract population densities. Metropolitan areas usually contain rural land that must be excluded to obtain the true population density and distribution. For example, the Ft Lauderdale, Fla, metropolitan area is coextensive with Broward County and includes uninhabited sections of the Everglades that should be excluded from consideration as part of the metropolitan area land base. The sprawl index is defined as

$$1) \qquad SI_i = 50((S\%_i - D\%_i) + 1),$$

where SI_i=sprawl index for metropolitan area, $S\%_i$=percentage of total population in low-density census tracts (>200 and <3500 persons per square mile), and $D\%_i$=percentage of the total population in high-density census tracts (≥3500 persons per square mile).

The index is transformed to a 0 to 100 scale (adding 1 to raw values converts scores to a 0–2 range, which is then multiplied by 50). Tracts were considered to be high density if they had a population density of 3500 or more persons per square mile and low density if they were below that threshold (3500 is the density at which people begin to use nonautomobile modes of transportation,[35] and it roughly divides the US metropolitan population into 2 equal halves). Tracts were considered rural and were excluded if the population density was fewer than 200 persons per square mile.

Sprawl index values were calculated for 330 metropolitan areas across the United States on the basis of the 2000 Census data. These areas had a mean sprawl index score of 68 (49 when weighted by population), which ranged from 3.94 to 100.[36] Other indexes of sprawl have been developed; however, many efforts to measure sprawl have relied on complex field surveys of individual metropolitan areas that (1) are too expensive to replicate nationally and thus limit their coverage to a subset of metropolitan areas,[37] (2) have been incompatible with other data sources,[38] or (3) have had methodological problems that reduce their utility for research.[39] Sprawl has been defined as a set of characteristics that include leapfrog-type development (development that often skips tracts closer to already developed areas in favor of more distant parcels, resulting in a pattern of developed land adjacent to undeveloped land), low density, employment dispersion, ugly architecture and design, automobile dependence, or other traits that are not easily and objectively measurable.[40] The measure used in this study was based on objectively developed census data (derived from published government data); it includes all metropolitan areas in the United States and is linear and normally distributed (Table 1).

TABLE 1—Selected Sprawl Index Values: US Census

Metropolitan Area	State	Sprawl Index
Atlanta	Ga	80.65
Augusta-Aiken	Ga	97.36
Boston–Worcester–Lawrence–Lowell–Brockton	Mass	55.84
Cedar Rapids	Iowa	70.69
Cincinnati	Ohio	61.64
Dallas	Tex	44.34
Dothan	Ala	100.00
Erie	Pa	57.01
Fort Collins–Loveland	Colo	51.77
Gainesville	Fla	76.61
Green Bay	Wis	65.88
Honolulu	Hawaii	35.46
Jacksonville	Fla	75.35
Lewiston–Auburn	Me	70.40
Los Angeles	Calif	10.61
Memphis	Tenn	62.78
Monroe	La	93.11
New York	NY	6.72
Orlando	Fla	64.34
Pittsburgh	Pa	57.74
Raleigh–Durham–Chapel Hill	NC	81.91
Rochester	NY	65.02
Salt Lake City–Ogden	Utah	34.80
Santa Rosa	Calif	55.44
Sioux Falls	SD	61.06
Tacoma	Wash	61.06
Tyler	Tex	86.32
West Palm Beach–Boca Raton	Fla	46.86
Youngstown	Ohio	77.05

Individual-Level Data

Several individual-level characteristics previously found to be related to the risk for being obese or overweight were included in this analysis: household income (<$10 000, $10 000–$14 999, $15 000–$19 999, $20 000–$24 999, $25 000–$34 999, $35 000–$49999, $50 000–$74 999, and ≥$75 000), education (kindergarten or never attended school, elementary education, some high school, high school graduate or GED, some college, or college graduate), and age (18–24 years, 25–34 years, 35–44 years, 45–54 years, 55–64 years, and ≥65 years). Dummy variables also associated with varying risks for being obese or overweight were included: Hispanic ethnicity, Black race, and female gender. For race/ethnicity, White race was the comparison group; for gender, male was the comparison group.

Analysis

Because the BRFSS uses a cluster sampling design, data were analyzed with Stata software, version 7.0 (Stata Corp, College Station, Tex), that incorporates measures to account for its sample design, weighting, and oversampling of certain populations. Weighting was used to avoid inaccurate point estimates of effect; strata and primary-sampling-unit vari-

TABLE 2—Number and Percentage of Overweight Individuals: Behavioral Risk Factor Surveillance System, 2000

	Number	Overweight, %	Obese, %
Total Sample	104 084	35.29	19.10
Race/ethnicity			
Black	10 596	36.37	28.44
Hispanic	13 198	36.01	23.23
White	77 137	35.30	17.66
Gender			
Male	50 241	44.66	20.19
Female	53 843	26.55	18.09

TABLE 3—Individual Variable Relative Risk for Overweight and Obesity: Behavioral Risk Factor Surveillance System, 2000

	Relative Risk (95% CI)	
	Overweight	Obese
Black (vs White)	1.41 (1.3, 1.53)	2.15 (1.98, 2.34)
Hispanic (vs White)	1.27 (1.16, 1.39)	1.56 (1.40, 1.74)
Female (vs male)	0.41 (.39, .43)	0.62 (.58, .65)
Age	1.01 (1.010, 1.013)	1.01 (1.010, 1.0135)
Income (per change in income category)	0.998 (.997, .998)	0.995 (.994, .996)
Education (per change in education category)	0.92 (.903, .943)	0.79 (.762, .804)
Sprawl index value (per 1-point change in index)	1.0007 (.9995, 1.0017)	1.0032 (1.002, 1.004)

Note. CI = confidence interval.

ables were incorporated into the analysis to obtain more accurate confidence intervals. Descriptive statistics were calculated first. Next, univariate logistic regression and multinomial logistic regression that used all independent variables (Taylor series methodology for both) analyzed the association between urban sprawl and being obese or overweight. The dependent variable was the respondent's calculated overweight or obese status, and the primary independent variable of interest was the sprawl index value of each respondent's metropolitan area of residence. Control variables were age, race/ethnicity, household income, and education, factors previously found to be associated with an increased risk for obesity.[41]

RESULTS

The final sample comprised 104 084 respondents, and similar to the BRFSS data, a large percentage of the sample was either overweight (35.3%) or obese (19.8%). Blacks, Hispanics, and males were more likely to be overweight or obese than the sample as a whole, and females were less likely to be overweight or obese (Table 2). In the univariate regression, where an individual's calculated obesity and overweight status were the dependent variables, individual-level characteristics previously found to be associated with increased risk for being obese or overweight also were associated with obesity and overweight status. Respondents who were Hispanic, Black, male, older, or had less household income or less education (age, income, and education were analyzed as continuous variables) were all more likely to be overweight or obese. When compared individually without controls for other explanatory variables, the association between urban sprawl and the risk for being overweight was small; however, the sprawl index was associated with an increased risk for being obese (Table 3).

When all control variables (sprawl index, female gender, Hispanic ethnicity, Black race, income, education, and age) were used in the multinomial logistic regression, the control variables again behaved as predicted: men, Hispanics, Blacks, low-income persons, less-educated persons, and older persons were all at increased risk for being both overweight and obese. In the adjusted analysis, the sprawl index score was associated with both increased risk for being overweight (0.2% for each 1-point increase in the sprawl index) and increased risk for being obese (0.5% for each 1-point increase in the sprawl index). The risk was greater for being obese than for being overweight (Table 4).

DISCUSSION

Urban sprawl was associated with an increased risk for being overweight or obese when individual variables were controlled. While the level of effect of a 1-point change in the sprawl index is small, the cumulative effects may be large because of the range of potential sprawl index values.

These findings should be interpreted with caution. While the BRFSS may generally be

TABLE 4—Full-Model Relative Risk for Overweight and Obesity: Behavioral Risk Factor Surveillance System, 2000

	Relative Risk (95% CI)	
	Overweight	Obese
Sprawl index value (per 1-point change in index)	1.002 (1.0006, 1.003)	1.005 (1.004, 1.006)
Female (vs male)	0.387 (.368, .405)	0.573 (.54, .61)
Hispanic (vs White)	1.436 (1.302, 1.584)	1.734 (1.55, 1.94)
Black (vs White)	1.618 (1.484, 1.764)	2.398 (2.20, 2.62)
Income (per change in income category)	0.997 (.996, .998)	0.994 (.993, .995)
Education (per change in education category)	0.946 (.924, .968)	0.826 (.80, .85)
Age	1.016 (1.01, 1.017)	1.016 (1.013, 1.017)

Note. CI =Confidence interval.

reliable, the validity of self-reported height and weight has been questioned because respondents tend to overreport height and underreport weight.[42,43] At least 1 study has found that reporting errors significantly affected obesity estimates.[44] Because of the multiple risk factors for obesity, applying this study's relative increased risk to individuals should be approached cautiously. However, if Atlanta (sprawl index=80.65) had the same level of sprawl as Boston (sprawl index=46.57), this model predicts that the risk for obesity in Atlanta adults would be reduced by approximately 17% after the demographic factors outlined in this study are controlled. There are no metropolitan estimates of obesity prevalence, but the CDC used the BRFSS to estimate statewide prevalence rates. In 2000, the CDC found the adult obesity prevalence rate in Georgia was 20.9%, which was approximately 27% higher than the Massachusetts adult obesity prevalence rate of 16.4%. These rates are not adjusted for demographic differences between the 2 states.[45]

This is a cross-sectional study. It may take years or decades to become overweight or obese, but the BRFSS only records the place of current residence. To the extent that respondents may move from metropolitan area to metropolitan area, and these areas have differing levels of urban sprawl, respondents' current place of residence may not reflect lifetime exposure to urban sprawl. Also, urban-sprawl levels have changed over time in individual metropolitan areas, although these changes have usually been modest. However, because most people do not change metropolitan areas or have lived in their current metropolitan area for a long period of time, and because urban sprawl levels are stable,[46] the current metropolitan-area urban-sprawl level may be an appropriate reflection of their exposure to urban sprawl. A longitudinal analysis of people's lifetime exposure to urban sprawl would clarify this issue.

While this semi-individual study analyzed individual- and metropolitan-level data together, the findings may still be an artifact of ecological bias. There are no data to reflect how urban sprawl may vary across a metropolitan area, or how urban sprawl may affect people differently. Another issue is that Blacks are more likely to live in the inner city[47,48] yet have higher rates of obesity and overweight status. This may mean that urban sprawl affects different people differently or that the effects of urban sprawl are limited to some groups. A related issue is that metropolitan areas are not homogenous but differ from inner city to older suburb to outer suburb. Urban sprawl may affect people living, working, or both in these different areas differently. A limitation of this study is that it did not control for this variety of neighborhood characteristics.

This study only includes noninstitutionalized metropolitan-dwelling US adults who lived in households with telephones and may not be generalizable to the entire adult population or to children. However, in 2000, 80% of the US population lived in metropolitan areas and 95% of households had telephones.[49] The exclusion of persons who lived outside metropolitan areas and those whose metropolitan area could not be determined could have affected the findings, but the obesity and overweight prevalence rates for the total sample were similar to the BRFSS data (Table 2). Similar studies that involve children would be appropriate because of the increasing rate of childhood obesity.

The causal association between urban sprawl and being obese or overweight could be in the reverse direction than is hypothesized here. People who are already overweight or obese may choose to move to metropolitan areas with greater levels of urban sprawl because they may find it easier to avoid walking or for other unknown reasons; however, there is no reason to believe this is the case.

Association is not equivalent to causation, although these findings are 1 piece of evidence of a link between urban sprawl and obesity. Perhaps urban sprawl affects the propensity to walk, bike, or be otherwise physically active. People in high-sprawled areas may drive more. It has been hypothesized that urban form may influence the mixture of transportation modes used by a population. The pattern of streets in a neighborhood may affect how people use their cars and their propensity to walk.[50,51] In an unpublished study by the author, metropolitan areas that had higher levels of urban sprawl had higher per capita vehicle miles traveled daily even after other factors, such as income, size of metropolitan area, and location in the southern United States, were controlled. A report by Smart Growth America used a subset of the same data and found a similar association between urban sprawl and driving.[52] This suggests 1 potential pathway of causality between urban sprawl and disease status: urban sprawl → increased automobile use → decreased physical activity → obesity → increased cardiovascular disease, diabetes, and other health problems.

Because there are multiple dimensions of urban sprawl and multiple ways of measuring it, the association between urban sprawl and the risk for obesity may vary by metric or characteristic. For example, the measure used in this study is based on the distribution of density. An urban-sprawl measure that is based on street connectivity—the degree to which blocks are small and walking between locations is possible—may demonstrate a different (or nonexistent) association. This analysis assumed a linear relationship between urban sprawl and risk for obesity, but this association might not be linear. Alternative associations, such as the possibility that urban-sprawl effects level off at very high and very low levels of urban sprawl, were not assessed.

Urban sprawl may reduce the amount of time available for physical activity because parks or fitness facilities are more distant. It also may affect diets by increasing distance to supermarkets or it may increase the cost of nutritious food by causing the conversion of farmland to urban uses.[53] The mechanisms of how urban sprawl might ultimately result in increased obesity need to be studied.

The causes of obesity are complex and involve diet, physical activity, and other factors. Foods high in fats or simple sugars, lifestyles that promote automobile use over walking, and perhaps yet unknown factors most likely interact to make people overweight or obese. This study did not address other important issues, such as the availability of fast food or how food insecurity may affect risk for being obese. Although some individual-level demographic characteristics were included in this analysis, other important factors, such as individual driving patterns, were not. The effects of these other factors might well be more important to the development of obe-

sity than urban sprawl is. Because urban sprawl might be an additional risk factor for obesity does not mean that attention to these causes of obesity should be ignored. It may be that urban sprawl interacts with other obesity risk factors.

CONCLUSIONS

This cross-sectional study found that higher levels of urban sprawl were associated with an increased risk for being overweight or obese among adults. If the association between urban sprawl and risk for being obese suggested by the results of this study is real, then urban sprawl may be contributing to a significant public health problem. Obesity takes a tremendous toll on people's health, and it costs the US economy billions of dollars. Because of the magnitude of these problems, the association between urban sprawl and risk for being obese warrants further attention. ■

About the Author

Russ Lopez is with the Department of Environmental Health, Boston University School of Public Health, Boston, Mass.

Requests for reprints should be sent to Russ Lopez, MCRP, DSc, Boston University School of Public Health, Department of Environmental Health, 715 Albany St, Talbott 2E, Boston, MA 02118 (e-mail: rptlopez@bu.edu).

This article was accepted April 1, 2003.

Acknowledgments

This study was funded by the National Institute of Environmental Health Sciences (NIEHS) (grant 5 F31 RS11219-02).

Note. The opinions expressed by this article are solely the responsibility of the author and do not necessarily represent the official views of the NIEHS or the National Institutes of Health.

Human Participant Protection

No human participants were included in this study.

References

1. Egede L, Zheng D. Modifiable cardiovascular risk factors in adults with diabetes: prevalence and missed opportunities for physician counseling. *Arch Inten Med.* 2002;162(4):427–433.

2. Wang G, Zheng Z, Heath G, Macera C, Pratt M, Buchner D. Economic burden of cardiovascular disease associated with excess body weight in US adults. *Am J Prev Med.* 2002;23(1):1–6.

3. Bianchini F, Kaaks R, Vainio H. Weight control and physical activity in cancer prevention. *Obes Rev.* 2002;3(1):5–8.

4. Solomon C, Manson J. Obesity and mortality: a review of the epidemiologic data. *Am J Clin Nutr.* 1997; 66(suppl 4):1044–1050.

5. Calle E, Thun M, Petrelli J, Rodriguez C, Heath C. Body-mass index and mortality in a prospective cohort of US adults. *N Engl J Med.* 1999;341(15):1097–1105.

6. Flegal K, Carroll M, Kuczmarski R, Johnson C. Overweight and obesity in the United States: prevalence and trends, 1960–1994. *Int J Obes Relat Metab Disord.* 1998;22:39–47.

7. Kuczmarski R, Flegal K, Campbell S, Johnson C. Increasing prevalence of overweight among US adults: the National Health and Nutrition Examination Surveys, 1960 to 1991. *JAMA.* 1994;272(3):205–211.

8. Flegal K, Carroll M, Ogden C, Johnson C. Prevalence and trends in obesity among US adults. *JAMA.* 2002;288(14):1723–1727.

9. Ogden C, Flegal K, Carroll M, Johnson C. Prevalence and trends in overweight among US children and adolescents, 1999–2000. *JAMA.* 2002;288(14): 1728–1732.

10. Mokdad A, Serdula M, Dietz W, Bauman B, Marks J, Koplan J. The spread of the obesity epidemic in the United States, 1991–1998. *JAMA.* 1999;282(16): 1519–1522.

11. Mokdad A, Bowman B, Ford E, Vinicor F, Marks J, Koplan J. The continuing epidemics of obesity and diabetes in the United States. *JAMA.* 2001;286(10): 1195–1200.

12. Downs A. *Some Realities About Sprawl and Urban Decline.* Washington, DC: Brookings Institute; 1999.

13. Duany A, Plater-Zyberk E, Speck J. *Suburban Nation.* New York, NY: North Point Press; 2000.

14. Jackson K. *Crabgrass Frontier: The Suburbanization of the United States.* New York, NY: Oxford University Press; 1985.

15. Kunstler J. *The Geography of Nowhere: The Rise and Decline of America's Man-Made Landscape.* New York, NY: Simon and Schuster; 1993.

16. Mieskowski P, Mills E. The causes of metropolitan suburbanization. *J Econ Perspect.* 1993;7(3):135–147.

17. Popenoe D. Urban sprawl: some neglected sociological considerations. *Sociol Soc Res.* 1979;63(2): 255–268.

18. Freeman L. The effects of sprawl on neighborhood social ties. An explanatory analysis. *J Am Plann Assoc.* 2001;67(1):69–77.

19. Berrigan D, Troiano R. The association between urban form and physical activity in US adults. *Am J Prev Med.* 2002;23(2S):74–79.

20. Craig C, Brownson R, Cragg S, Dunn A. Exploring the effect of the environment on physical activity. A study examining walking to work. *Am J Prev Med.* 2002;23(2S):36–43.

21. Giles-Corti B, Giles-Corti R. The relative influence of individual, social and physical environment determinants of physical activity. *Soc Sci Med.* 2002;54: 1793–1812.

22. Jandy S, Boarnet M, Ewing R, Killingsworth R. How the built environment affects physical activity. Views from urban planning. *Am J Prev Med.* 2002; 23(2S):64–73.

23. Poston W, Foreyt J. Obesity is an environmental issue. *Atheroslcerosis.* 1999;146:201–209.

24. Jackson R, Kochtitzky C. *Creating a Healthy Environment: The Impact of the Built Environment on Public Health.* Washington DC: Sprawl Watch Clearinghouse; 2002.

25. Pierce N. Fight Fat with Well-Planned Communities. *The Charlotte Observer.* April 28, 2001:21.

26. Kreling C. Fat city. *Planning.* 2001(June):4–9.

27. O'Toole R. Special interests run with faulty obesity data. *Cascade Commentary.* 2002;9:1.

28. Diez-Roux A. Bringing context back into epidemiology: variables and fallacies in multilevel analysis. *Am J Public Health.* 1998;88:216–222.

29. Von Korff M, Koepsell T, Curry S, Diehr P. Multilevel analysis in epidemiological research on health behaviors and outcomes. *Am J Epidemiol.* 1992;135: 1077–1082.

30. Duncan C, Jones K, Moon G. Health-related behaviour in context: a multilevel medalling approach. *Soc Sci Med.* 1996;42(6):817–830.

31. Centers for Disease Control and Prevention (CDC). *BRFSS–Frequently Asked Questions.* Available at: http://www.cdc.gov/brfss/faqs.htm#3. Accessed March 20, 2003.

32. CDC. *Behavioral Risk Factor Surveillance System Survey Data.* Atlanta, Ga: US Dept of Health and Human Services, CDC: 2000.

33. National Institutes of Health (NIH). *Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults.* Bethesda, Md: Dept of Health and Human Services, NIH, National Heart, Lung, and Blood Institute: 1998.

34. Willett W, Dietz W, Colditz G. Primary care: guidelines for healthy weight. *N Engl J Med.* 1999; 341(6):427–434.

35. Ross C, Dunning A. *Land Use Transportation Interaction: An Examination of the 1995 NPTS Data.* Washington, DC: Federal Highway Administration: 1997.

36. Lopez R, Hynes HP. Sprawl in the 1990s: measurement, distribution and trends. *Urban Aff Rev.* 2003;38(3):325–355.

37. Galster G, Hanson R, Woman H, Coleman S, Frirebage J. *Wrestling Sprawl to the Ground: Defining and Measuring an Elusive Concept.* Washington, DC: Fannie Mae Foundation; 2000.

38. Fulton W, Pendall R, Nguyen M, Harrison A. *Who Sprawls Most? How Growth Patterns Differ Across the US.* Washington, DC: Brookings Institute; 2001.

39. Glaeser E, Kahn M. Decentralized employment and the transformation of the American city. 2001. *Brookings-Wharton Papers on Urban Affairs*: Washington, DC.

40. Beaumont C. *How Superstore Sprawl Can Harm Communities: And What Citizens Can Do About It.* Washington, DC: National Trust for Historic Preservation; 1997.

41. Paeratukui S, Lovejoy J, Ryan D, Bray G. The relation of gender, race and socioeconomic status to obesity and comorbidities in a sample of US adults. *Int J Obes Relat Metab Disord.* 2002;26(9):1205–1210.

42. Bowlin S, Morrill B, Nafziger A, Jenkins P, Lewis C, Pearson T. Validity of cardiovascular disease risk factors assessed by telephone survey: the Behavioral Risk Factor Survey. *J Clin Epidemiol.* 1993;46(6):561–571.

43. Jackson C, Jatulis D, Fortmann S. The Behavioral Risk Factor Survey and the Stanford Five-City Project Survey: a comparison of cardiovascular risk behavior estimates. *Am J Public Health.* 1992;82(3):412–416.

44. Lakdawalla D, Philipson T. *The Growth of Obesity and Technological Change: A Theoretical and Empirical Examination.* Cambridge, Mass: National Bureau of Economic Research; 2002..

45. CDC. *1991–2001 Prevalence of Obesity Among US Adults by State.* Atlanta, Ga: CDC; 2002.

46. Sheehan M. *City Limits. Putting the Brake on Sprawl.* Washington, DC: Worldwatch Institute; 2001.

47. Farley R, Schuman H, Bianchi S, Colasant K, Hatchett S. "Chocolate city, vanilla suburbs": will the trend toward racially separate communities continue? *Soc Sci Res.* 1978;7(4):319–344.

48. Farley R. Continued racial residential segregation in Detroit: "Chocolate city, vanilla suburbs": revisited. *J Housing Res.* 1993;4(1):1–38.

49. US Bureau of the Census. *March 2001 Current Population Survey.* Washington, DC: US Bureau of the Census; 2001.

50. Boarnet M, Randall C. The influence of land use on travel behavior: specification and estimation strategies. *Transportation Res Part A: Policy Pract.* 2001;35(9): 823–845.

51. Srinivasan S, Ferreira J. Travel behavior at the household level: understanding linkages with residential choice. *Transportation Res Part D: Transport Environ.* 2002;7(3):225–242.

52. Ewing R, Pendall R, Chen D. *Measuring Sprawl and Its Impact.* Washington, DC: Smart Growth America; 2002.

53. Frumkin H. Urban sprawl and public health. *Public Health Rep.* 2002;117:201–217.

ISBN 0-87553-030-3
softcover ▮ 2004
$27.50 APHA Members
$35.95 Nonmembers
plus shipping and handling

NEW!

Race and Research
Perspectives on Minority Participation in Health Studies

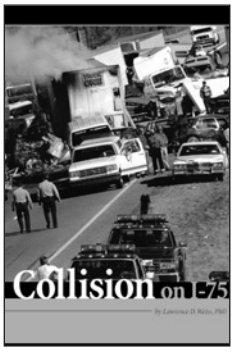
Edited by Bettina Beech, DrPH, MPH, and Maurine Goodman, MA, MPH

Race and Research: Perspectives on Minority Participation in Health Studies is a teaching text and resource guide for students, health professionals, public health researchers, and the general public that extends the discussion of environmental factors that influence ethnic minority participation in health studies. This book examines the lack of minority participation in health studies from social, historical, and scientific perspectives.

This book is divided into three main sections: 1) The Meaning of Race, Culture and Ethnicity in Research; 2) Health Studies and Ethnic Minority Populations and 3) The Impact of Revolutionary Changes in Medicine and Health Care on Minority Participation in Health Studies.

American Public Health Association
Publication Sales
Web: www.apha.org
E-mail: APHA@TASCO1.com
Tel: (301) 893-1894
FAX: (301) 843-0159

RR12J1



ISBN 0-87553-032-X
2004 ▮ softcover ▮ 125 pages
$19.95 APHA Members
$26.96 Nonmembers
plus shipping and handling

NEW from APHA

Collision on I-75
by Lawrence D. Weiss, PhD

Collision on I-75 tells a compelling public health story that has not been told before. It details two decades of struggle by public health professionals, legislators, state officials, and law enforcement to compel a large corporation to prevent deadly, industrial-fog–related traffic accidents.

In December 1990, nearly one hundred vehicles collided on Interstate-75 in Tennessee in an unusually dense fog bank, leaving 12 dead and dozens seriously injured. One attorney led a lawsuit on behalf of most of the victims and found that the cause of the massive collision was industrial pollution produced by a pulp mill north of the collision site.

This is the true story of an incident involving corporate negligence, faulty state regulation, and a risk-taking attorney in pursuit of uncertain compensation for the victims and himself.

American Public Health Association
Publication Sales
Web: www.apha.org
E-mail: APHA@TASCO1.com
Tel: (301) 893-1894
FAX: (301) 843-0159

C17504J3